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DATE: Sunday, May 22, 2005

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		DB=USPT; PLUR=YES; OP=AND	
☐	L1	method.clm. same (thioredoxin or thio-redoxin or trx).clm.	56
☐	L2	(thioredoxin or thio-redoxin or trx) near10 (administer$ or treating or treatment or therapeutic or therapeutically or preventing)	68
☐	L3	L2 and l1	18
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=AND	
☐	L4	(windel or kellehor or $toole).in.	1136
☐	L5	L4 and (trx ir thio or thioredoxin or thio-redox or thioredoxin)	5
☐	L6	L4 and (trx or thio or thioredoxin or thio-redox or thioredoxin)	8
☐	L7	(trx or thio or thioredoxin or thio-redox or thioredoxin).ti,ab.	27769
☐	L8	(trx or thioredoxin or thio-redox or thioredoxin).ti,ab.	617
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END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 50 of 56 returned.

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Terms	Documents
method.clm. same (thioredoxin or thio-redoxin or trx).clm.	56

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Entry 7 of 56 File: USPT Feb 10, 2004

US-PAT-NO: 6689775
DOCUMENT-IDENTIFIER: US 6689775 B2

TITLE: Uses of thioredoxin

DATE-ISSUED: February 10, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Powis; Garth	Tuscon	AZ		

US-CL-CURRENT: 514/208; 424/9.1, 424/9.361, 435/7.1, 514/183

CLAIMS:

I claim:

1. A method of inhibiting tumor growth in vivo in a tumor cell that over-expresses thioredoxin comprising contacting said tumor cell with a cell growth inhibiting effective amount of an inhibitor of thioredoxin, said inhibitor of thioredoxin interacting with a cysteine of human thioredoxin at residue 73 of said human thioredoxin.

2. The method of claim 1, wherein said inhibitor is a 2-imidazolyl disulfide.

3. The method in claim 2, wherein said inhibitor of thioredoxin expression binds the cysteine at residue 73 of human thioredoxin.

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L1: Entry 8 of 56 File: USPT Jan 13, 2004

US-PAT-NO: 6677433
DOCUMENT-IDENTIFIER: US 6677433 B2

TITLE: Stabilization of hypoallergenic, hyperdigestible previously reduced proteins

DATE-ISSUED: January 13, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Buchanan; Bob B.	Berkeley	CA		
Morigasaki; Susumu	Berkeley	CA		
del Val; Gregorio	San Diego	CA		
Frick; Oscar L.	San Francisco	CA		

US-CL-CURRENT: 530/350; 424/94.4, 426/34, 426/541, 426/549, 426/574, 426/656, 530/345, 530/365, 530/370, 530/378

CLAIMS:

What is claimed is:

1. A method of producing an allergenic protein with reduced allergenicity comprising: (a) treating an allergenic protein containing disulfide bonds with an amount of thioredoxin, nicotinamide adenine dinucleotide phosphate-thioredoxin reductase and NADPH, or an amount of dithiothreitol effective to and at a temperature effective to reduce at least one disulfide bond in said allergenic protein; and (b) reacting the allergenic protein from step (a) with an amount of a physiological disulfide effective to minimize reoxidation of at least one reduced disulfide bonds in said allergenic protein from step (a).

2. The method of claim 1 wherein the allergenic protein is selected from the group consisting of cow's milk, egg, soy, rice, wheat, barley, peanut and pollen proteins.

3. The method of claim 1 wherein the physiological disulfide is cystamine or oxidized glutathione.

4. The method of claim 3 wherein the amount of cystamine is about 0.4 .mu.moles to about 40 .mu.moles per mg of protein.

5. The method of claim 3 wherein the amount of oxidized glutathione is about 0.4 .mu.moles to about 40 .mu.moles per mg of protein.

6. A method of producing an allergenic food with reduced allergenicity comprising: (a) contacting an allergenic food having at least one allergenic protein containing disulfide bonds with an amount of thioredoxin, nicotinamide adenine dinucleotide phosphate-thioredoxin reductase and NADPH effective to reduce at least one disulfide bond in said protein; and (b) treating the food from step (a) with an amount of a physiological disulfide effective to

minimize reoxidation of at least one reduced disulfide bond in said protein in said food of step (a).

7. The method of claim 6 wherein said food contains milk or peanut.

8. The method of claim 6 wherein the physiological disulfide is cystamine or oxidized glutathione.

9. The method of claim 8 wherein the amount of cystamine is about 0.4 .mu.moles to about 40 .mu.moles per mg of protein in said food.

10. The method of claim 8 wherein the amount of oxidized glutathione is about 0.4 .mu.moles to about 40 .mu.moles per mg of protein in said food.

11. A method of producing an allergenic protein with reduced allergenicity comprising: (a) reducing an allergenic protein containing disulfide bonds wit an amount of thioredoxin, nicotinamide adenine dinucleotide phosphate-thioredoxin reductase and NADPH, or dithiothreitol, or lipoic acid effective to reduce said allergenic protein; and (b) reacting the allergenic protein from step (a) with an amount of a physiological disulfide effective to stabilize said allergenic protein from step (a).

12. The method of claim 11 wherein the allergenic protein is selected from the group consisting of cow's milk, egg, soy, rice, wheat, barley, peanut and pollen proteins.

13. The method of claim 11 wherein the physiological disulfide is cystamine or oxidized glutathione.

14. A method of decreasing an allergic reaction caused by an allergenic protein in an animal comprising administering to the animal an amount of the protein with reduced allergenicity effective to reduce the allergic reaction, wherein the allergenic protein with reduced allergenicity has at least one disulfide bond reduced by thioredoxin, nicotinamide adenine dinucleotide phosphate-thioredoxin reductase and NADPH, or dithiothreitol, and subsequently reacted with a physiological disulfide for a time and under conditions where reoxidation of at least one reduced disulfide bond is minimized.

15. A method of decreasing an allergic reaction caused by an allergenic food having at least one allergenic protein in an animal comprising administering to the animal an amount of the food with reduced allergenicity effective to reduce the allergic reaction, wherein the allergenic protein in the food with reduced allergenicity has at least one disulfide bond reduced by thioredoxin, nicotinamide adenine dinucleotide phosphate-thioredoxin reductase and NADPH, and subsequently reacted with a physiological disulfide for a time and under conditions where reoxidation of at least one reduced disulfide bond is minimized.

16. A method of decreasing an allergic reaction caused by an allergenic protein in an animal comprising administering to the animal an amount of the protein with reduced allergenicity effective to reduce the allergic reaction, wherein the allergenic protein with reduced allergenicity has at least one disulfide bond reduced by thioredoxin, nicotinamide adenine dinucleotide phosphate-thioredoxin reductase and NADPH, or dithiothreitol, or lipoic acid, and subsequently reacted with a physiological disulfide for a time and under conditions where said allergenic protein with reduced allergenicity is stabilized.

US-PAT-NO: 6605278
DOCUMENT-IDENTIFIER: US 6605278 B1
**** See image for Certificate of Correction ****

TITLE: Uses of trank, a novel secretory cytokine

DATE-ISSUED: August 12, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Aggarwal; Bharat B.	Houston	TX		

US-CL-CURRENT: 424/145.1; 514/2, 530/387.9, 530/389.2

CLAIMS:

What is claimed is:

1. A method of inhibiting TRANK-mediated NF-.kappa.B activation in an individual in need of such treatment, comprising the step of administering an effective dose of an anti-TRANK antibody to said individual, wherein said antibody reacts with human TRANK (thioredoxin peroxidase-related activator of NF-.kappa.B and c-Jun N-terminal kinase) protein of SEQ ID No. 1, and wherein binding of said antibody to said TRANK protein inhibits TRANK-mediated activation of NF-.kappa.B.

2. A method for treating a pathophysiological state in a human wherein said state has an undesirable level of TRANK-mediated NF-.kappa.B activation, comprising the step of administering to said human an effective dose of an anti-TRANK antibody, wherein said antibody reacts with human TRANK (thioredoxin peroxidase-related activator of NF-.kappa.B and c-Jun N-terminal kinase) protein of SEQ ID No. 1, and wherein binding of said antibody to said TRANK protein inhibits TRANK-mediated activation of NF-.kappa.B.

3. The method of claim 2, wherein said pathological state is toxic shock, septic shock, acute phase response, viral infection, radiation susceptibility, atherosclerosis, cancer, acute inflammatory conditions or graft vs. host reaction.

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First Hit Fwd Refs

L1: Entry 23 of 56 File: USPT Jun 18, 2002

DOCUMENT-IDENTIFIER: US 6406701 B1
TITLE: Method and compositions for preventing or reducing HIV infection

CLAIMS:

3. The method according to claim 2 wherein the thiol containing compound is thioredoxin.

9. The method according to claim 8 wherein the thiol containing compound is selected from the group consisting of thioredoxin and glutathione.

19. The method according to claim 18 wherein the thiol containing compound is thioredoxin.

24. The method according to claim 23 wherein the thiol containing compound is thioredoxin.

First Hit Fwd Refs

L1: Entry 39 of 56 File: USPT Nov 16, 1999

US-PAT-NO: 5985261
DOCUMENT-IDENTIFIER: US 5985261 A

TITLE: Use of thioredoxin-like molecules for induction of MnSOD to treat oxidative damage

DATE-ISSUED: November 16, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
White; Carl W.	Denver	CO		
Das; Kumuda C.	Aurora	CO		

US-CL-CURRENT: 424/85.1; 424/450, 424/85.2, 514/12, 514/17, 514/18, 514/2, 514/824, 514/826, 514/886, 530/329, 530/330, 530/399

Full - [FULL]
Title - [TI]
Citation - [CIT]
Front - [FRO]
Review - [REV]
Classification - [CLS]
Date - [DATE]
Reference - [REF]
Sequences - [SEQ]
Attachments - [ATT]
Claims - [CLM]
KWIC - [KWIC]
Drwg Desc - [DRAW]
Image - [IMG]

CLAIMS:

What is claimed is:

1. A method to increase a cellular level of manganese superoxide dismutase (MnSOD) in an animal to treat oxidative damage associated with a lung disease, comprising administering to lung tissue of said animal an amount of a protein containing a thioredoxin active-site in reduced state effective to induce the production of MnSOD.

2. The method of claim 1, wherein said lung disease is selected from the group consisting of infant respiratory distress syndrome, adult respiratory distress syndrome, interstitial lung disease, and asthma.

3. The method of claim 1, wherein said protein is administered by at least one route selected from the group consisting of nasal, intratracheal and inhaled routes.

4. The method of claim 1, wherein said protein is administered in a pharmaceutically acceptable delivery vehicle.

5. The method of claim 1, wherein said protein is administered in an amount that is between about 1.5 mmoles/kg and about 150 mmoles/kg.

6. The method of claim 1, wherein said protein is administered in an amount that is between about 2 mmoles/kg and about 25 mmoles/kg.

7. The method of claim 1, wherein said protein is administered in an amount that is between about 3 mmoles/kg and about 10 mmoles/kg.

8. The method of claim 1, wherein said protein has a half-life in said animal of between about 5 minutes and about 24 hours.

9. The method of claim 1, wherein said protein has a half-life in said animal of between about 2 hours and about 16 hours.

10. The method of claim 1, wherein said protein has a half-life in said animal of between about 4 hours and about 12 hours.

11. The method of claim 1, wherein said cellular level of MnSOD is increased at least about 2-fold.

12. The method of claim 1, wherein said cellular level of MnSOD is increased at least about 5-fold.

13. The method of claim 1, wherein said cellular level of MnSOD is increased at least about 10-fold.

14. The method of claim 1, wherein said thioredoxin active-site comprises the amino acid sequence C-X-X-C (SEQ ID NO:1), wherein C residues are in reduced state, and wherein X residues are any amino acid residue.

15. The method of claim 1, wherein said thioredoxin active-site comprises the amino acid sequence X-C-X-X-C-X (SEQ ID NO:3), wherein C residues are in reduced state, and wherein X residues are any amino acid residue.

16. The method of claim 1, wherein said thioredoxin active-site comprises the amino acid sequence X-C-G-P-C-X (SEQ ID NO:4), wherein C residues are in reduced state, and wherein X residues are any amino acid residue.

17. The method of claim 1, wherein said thioredoxin active-site comprises the amino acid sequence W-C-G-P-C-K (SEQ ID NO:5), wherein C residues are in reduced state.

18. The method of claim 1, wherein said protein comprises thioredoxin selected from the group consisting of prokaryotic thioredoxin, yeast thioredoxin and mammalian thioredoxin.

19. The method of claim 1, wherein said protein comprises human thioredoxin.

20. The method of claim 1, wherein said animal is a mammal.

21. The method of claim 20, wherein said mammal is a human.

22. A composition for increasing a cellular level of MnSOD in an animal to treat oxidative damage associated with a lung disease, comprising a protein containing a thioredoxin active-site in reduced state effective to induce the production of MnSOD, formulated with a pharmaceutically acceptable delivery vehicle selected from the group consisting of liposomes, lipospheres and surfactants, which provides a half-life of between about 5 minutes and about 24 hours in said animal.

23. The composition of claim 22, wherein said thioredoxin active-site comprises the amino acid sequence C-X-X-C (SEQ ID NO:1), wherein C residues are in reduced state, and wherein said X residues are any amino acid residue.

24. The composition of claim 22, wherein said thioredoxin active-site comprises the amino acid sequence X-C-X-X-C-X (SEQ ID NO:3), wherein C

residues are in reduced state, and wherein said X residues are any amino acid residue.

25. The composition of claim 22, wherein said thioredoxin active-site comprises the amino acid sequence X-C-G-P-C-X (SEQ ID NO:4), wherein C residues are in reduced state, and wherein said X residues are any amino acid residue.

26. The composition of claim 22, wherein said thioredoxin active-site comprises the amino acid sequence W-C-G-P-C-K (SEQ ID NO:5), wherein C residues are in reduced state.

27. The composition of claim 22, wherein said formulation further comprises about 0.1 unit/liter achieved surface concentration of thioredoxin reductase and about 1 mM achieved surface concentration of nicotinamide-adenine dinucleotide phosphate (reduced form) (NADPH).

28. The composition of claim 22, wherein said protein comprises thioredoxin selected from a group consisting of prokaryotic thioredoxin, yeast thioredoxin and mammalian thioredoxin.

29. The composition of claim 22, wherein said protein comprises human thioredoxin.

30. A method to treat an animal from cellular injury due to oxidative damage associated with a lung disease by increasing a cellular level of manganese superoxide dismutase (MnSOD), comprising administering to lung tissue of said animal a protein comprising the amino acid sequence X-C-X-X-C-X (SEQ ID NO:3), wherein C residues are in reduced state, effective to induce the production of MnSOD.

31. The method of claim 30, wherein said protein comprises the amino acid sequence X-C-G-P-C-X (SEQ ID NO:4).

32. The method of claim 30, wherein said protein comprises the amino acid sequence W-C-G-P-C-K (SEQ ID NO:5).

33. The method of claim 30, wherein said protein has a half-life in the animal of between about 5 minutes and about 24 hours.

34. The method of claim 30, wherein said protein is administered in an amount that is between about 1.5 mmoles/kg and about 150 mmoles/kg.

35. The method of claim 30, wherein said protein comprises thioredoxin selected from a group consisting of prokaryotic thioredoxin, yeast thioredoxin and mammalian thioredoxin.

36. The method of claim 30, wherein said lung disease is selected from the group consisting of infant respiratory distress syndrome, adult respiratory distress syndrome, interstitial lung disease, and asthma.

37. The method of claim 30, wherein said animal is a human.

20030223980. 20 Jun 03. 04 Dec 03. Uses of thioredoxin. Powis, Garth. 424/94.4; 435/7.23 A61K038/44 G01N033/574.

02369178 97407635 PMID: 9264384

Interleukin-15 + thioredoxin induce DNA synthesis in B-chronic lymphocytic leukemia cells but not in normal B cells.

Soderberg O; Christiansen I; Nilsson G; Carlsson M; Nilsson K

Department of Pathology, University of Uppsala, University Hospital, Sweden.

Leukemia (ENGLAND) Aug 1997, 11 (8) p1298-304, ISSN 0887-6924 Journal Code: 8704895

Document Type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

We have previously shown that Staphylococcus aureus Cowan strain 1 particles (SAC) + thioredoxin (Trx) + IL-2 may induce B-chronic lymphocytic leukemia (B-CLL) cells to proliferate. In this paper we have examined IL-15, which has activities similar to IL-2, for its ability to stimulate B-CLL cells and compared its activity with that of IL-2. We found that B-CLL cells could be induced to DNA synthesis upon treatment with IL-15 + Trx. The presence of Trx was essential for the IL-15-induced DNA synthesis. This contrasts to the effect of IL-15 + Trx on normal CD5+ and CD5- B cells, where IL-15 + Trx alone only induced limited DNA synthesis. IL-15 was as effective in the induction of DNA synthesis in B-CLL cells as IL-2, but about 100-fold less potent with an EC50 of 200 ng/ml. In addition we found that the IL-15 + Trx-induced proliferation was inhibited by CD40 stimulation. We conclude that IL-15 together with a proper costimulus can induce B-CLL cells to proliferate in vitro.

Tags: Human; Support, Non-U.S. Gov't

Major Descriptors: *B-Lymphocytes--cytology--CY; *DNA--biosynthesis--BI; *Interleukin-15--administration and dosage--AD; *Leukemia, B-Cell, Chronic --pathology--PA; * **Thioredoxin** --administration and dosage--AD

Minor Descriptors: Antigens, CD40--physiology--PH; Cell Cycle --drug effects--DE; Cell Differentiation--drug effects--DE; Cell Division--drug effects--DE; Lymphocyte Transformation; Receptors, Interleukin-2 --metabolism--ME; Tumor Cells, Cultured

CAS Registry No.: 0 (Antigens, CD40); 0 (Interleukin-15); 0 (Receptors, Interleukin-2); 52500-60-4 (Thioredoxin); 9007-49-2 (DNA)

Record Date Created: 19970904

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E33       30       THIOREDOXIN REDUCTASE (NADPH) --ANALYSIS --AN
E34       67       THIOREDOXIN REDUCTASE (NADPH) --ANTAGONISTS AN
E35       38       THIOREDOXIN REDUCTASE (NADPH) --BIOSYNTHESIS -
E36        7       THIOREDOXIN REDUCTASE (NADPH) --BLOOD --BL

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Set     Items   Description
S1         35   'THIOREDOXIN --ADMINISTRATION AND DOSAGE --AD' OR 'THIORED-
              OXIN --DRUG EFFECTS --DE' OR 'THIOREDOXIN --PHYSIOLOGY --PH' -
              OR 'THIOREDOXIN --THERAPEUTIC USE --TU'
? s s1/2001:2005
              35  S1
         2442758  PY=2001 : PY=2005
      S2      12  S1/2001:2005
? s s1 not s2
              35  S1
              12  S2
      S3      23  S1 NOT S2
? t s3/9/all

 3/9/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2005 The Dialog Corp. All rts. reserv.

13044623   PMID: 11006259
  Lipid peroxidation and peroxynitrite in retinal ischemia-reperfusion injury.
  Shibuki H; Katai N; Yodoi J; Uchida K; Yoshimura N
  Department of Ophthalmology, Shinshu University School of Medicine, Matsumoto, Japan.
  Investigative ophthalmology & visual science (UNITED STATES)  Oct 2000, 41 (11) p3607-14, ISSN 0146-0404  Journal Code: 7703701
  Publishing Model Print

  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  Subfile:  INDEX MEDICUS
  PURPOSE: To investigate whether lipid peroxides play a role in retinal cell death due to ischemia-reperfusion injury, whether recombinant human thioredoxin (rhTRX) treatment reduces production of lipid peroxides of the retina, and whether such treatment reduces the number of cells expressing c-Jun and cyclin D1. METHODS: Retinal ischemia was induced in rats by increasing the intraocular pressure to 110 mm Hg for 60 minutes. After reperfusion, immunohistochemical staining for lipid peroxide, peroxynitrite, c-Jun, and cyclin D1 and propidium iodide (PI) staining were performed on retinal sections from animals treated intravenously with and without rhTRX, a free radical scavenger. Quantitative analyses of PI-, c-Jun-, and cyclin D1-positive cells were performed after the ischemic insult. Concentration of lipid peroxides in the retina was determined by the thiobarbituric acid assay. RESULTS: Specific immunostaining for lipid peroxides was seen in the ganglion cell layer at 6 hours after reperfusion, in the inner nuclear layer at 12 hours, and in the outer nuclear layer at 48 hours. Time course studies for PI-positive cells in the three nuclear layers coincided with those of specific immunostaining for lipid peroxides. The specific immunostaining was weakened by pre- and posttreatment with 0.5
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mg of rhTRX. The number of PI-, c-Jun-, and cyclin D1-positive cells and the concentration of lipid peroxides were significantly decreased by treatment with rhTRX compared with those of vehicle-treated control rats (P: < 0. 01). CONCLUSIONS: Lipid peroxides formed by free radicals may play a role in neuronal cell death in retinal ischemia-reperfusion injury.

Tags: Male; Research Support, Non-U.S. Gov't

Descriptors: *Lipid Peroxidation; *Lipid Peroxides--metabolism--ME; *Nitrates--metabolism--ME; *Reperfusion Injury--metabolism--ME; *Retina --metabolism--ME; *Retinal Diseases--metabolism--ME; Aldehydes--metabolism --ME; Animals; Cell Death; Cyclin D1--metabolism--ME; Fluorescent Antibody Technique, Indirect; Free Radical Scavengers--therapeutic use--TU; Propidium--metabolism--ME; Proto-Oncogene Proteins c-jun--metabolism--ME; Rats; Rats, Sprague-Dawley; Recombinant Proteins--therapeutic use--TU; Reperfusion Injury--drug therapy--DT; Reperfusion Injury--pathology--PA; Retina--pathology--PA; Retinal Diseases--drug therapy--DT; Retinal Diseases--pathology--PA; Thiobarbituric Acid Reactive Substances; **Thioredoxin** --therapeutic use--TU

CAS Registry No.: 0 (Aldehydes); 0 (Free Radical Scavengers); 0 (Lipid Peroxides); 0 (Nitrates); 0 (Proto-Oncogene Proteins c-jun); 0 (Recombinant Proteins); 0 (Thiobarbituric Acid Reactive Substances); 136601-57-5 (Cyclin D1); 26404-66-0 (peroxynitric acid); 29343-52-0 (4-hydroxy-2-nonenal); 36015-30-2 (Propidium); 52500-60-4 (Thioredoxin)

Record Date Created: 20001012

Record Date Completed: 20001012

3/9/2

DIALOG(R)File 155:MEDLINE(R)

12977419 PMID: 10930459

Thioredoxin peroxidase is required for the transcriptional response to oxidative stress in budding yeast.

Ross S J; Findlay V J; Malakasi P; Morgan B A

School of Biochemistry and Genetics, The Medical School, University of Newcastle upon Tyne, United Kingdom.

Molecular biology of the cell (UNITED STATES) Aug 2000, 11 (8) p2631-42, ISSN 1059-1524 Journal Code: 9201390

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

A genetic screen was performed in Saccharomyces cerevisiae to identify mechanisms important for the transcriptional activation of genes encoding antioxidant proteins. Thioredoxin peroxidase, Tsa1p, of the thioredoxin system, was found to be essential for the transcriptional induction of other components of the thioredoxin system, TRX2 (thioredoxin) and TRR1 (thioredoxin reductase), in response to H(2)O(2). The expression of TRX2 and TRR1 is known to be regulated by the transcription factors Yap1p and Skn7p in response to H(2)O(2), and the Tsa1p-dependent regulation of TRX2 requires the Yap1p/Skn7p pathway. The data suggest that expression of components of the thioredoxin system is dependent on the activity of Tsa1p in response to H(2)O(2) in a Yap1p/Skn7p-dependent pathway.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *DNA-Binding Proteins--physiology--PH; *Hydrogen Peroxide --pharmacology--PD; *Oxidative Stress; *Peroxidases--physiology--PH; *Saccharomyces cerevisiae--physiology--PH; *Saccharomyces cerevisiae

Proteins; *Thioredoxin--metabolism--ME; *Thioredoxin Reductase (NADPH) --metabolism--ME; *Transcription Factors--physiology--PH; Animals; Catalase --genetics--GE; Catalase--metabolism--ME; DNA-Binding Proteins--genetics --GE; Gene Deletion; Gene Expression Regulation; Models, Biological; Peroxidases--drug effects--DE; Peroxidases--genetics--GE; Point Mutation; Saccharomyces cerevisiae--drug effects--DE; Saccharomyces cerevisiae --genetics--GE; Signal Transduction; **Thioredoxin** --drug effects--DE; Thioredoxin--genetics--GE; Thioredoxin Reductase (NADPH)--drug effects--DE ; Thioredoxin Reductase (NADPH)--genetics--GE; Transcription Factors --genetics--GE; Transcription, Genetic--drug effects--DE

CAS Registry No.: 0 (DNA-Binding Proteins); 0 (SKN7 protein, S cerevisiae); 0 (Saccharomyces cerevisiae Proteins); 0 (Transcription Factors); 139046-61-0 (YAP1 protein, S cerevisiae); 52500-60-4 (Thioredoxin); 7722-84-1 (Hydrogen Peroxide)

Enzyme No.: EC 1.11.1. (Peroxidases); EC 1.11.1.- (PRDX3 protein, human); EC 1.11.1.- (protector protein (mixed-function oxidase systems)); EC 1.11.1.6 (Catalase); EC 1.6.4.5 (Thioredoxin Reductase (NADPH))

Record Date Created: 20000905

Record Date Completed: 20001222

3/9/3

DIALOG(R)File 155:MEDLINE(R)

12108788 PMID: 9406241

Redox active disulfides: the thioredoxin system as a drug target.

Kirkpatrick D L; Ehrmantraut G; Stettner S; Kunkel M; Powis G

Department of Chemistry, University of Regina, Canada. kirkpaly@leroy.cc.uregina.ca

Oncology research (UNITED STATES) 1997, 9 (6-7) p351-6, ISSN 0965-0407 Journal Code: 9208097

Contract/Grant No.: CA 48725; CA; NCI

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Thioredoxin, and particularly extracellular thioredoxin, presents an attractive target for developing novel agents to treat cancer. Our studies have involved the examination of a series of alkyl 2-imidazolyl disulfides as inhibitors of the growth-stimulatory activity of the thioredoxin system. We originally determined the disulfides to be weak reversible inhibitors of thioredoxin reductase. Subsequently, we have shown that alkyl 2-imidazolyl disulfides interact directly with thioredoxin, thioalkylating critical cysteine residues or causing dimerization of the protein leading to its loss of biological activity. One of the analogues that binds to thioredoxin, 1-methylpropyl 2-imidazolyl disulfide (IV-2), selectively inhibits the thioredoxin-dependent growth of tumor cells in culture and has antitumor activity against MCF-7 and HL-60 tumors in vivo. Our work involves the development of a parallel combinatorial synthetic method to produce a large number of disulfide analogues at one time. These analogues, which differ sterically, electronically, and physically, were produced in a 96-well plate. The biological activity of these analogues was evaluated, also in the 96-well plate format. This rapid method of evaluating biological activity is a means to identify agents with specificity for inhibition of the thioredoxin system, and may provide novel antitumor agents with activity against solid tumor cancers.

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Descriptors: *Antineoplastic Agents--pharmacology--PD; *Disulfides --pharmacology--PD; * **Thioredoxin** --drug effects--DE; *Thioredoxin --metabolism--ME; Animals; Apoptosis--drug effects--DE; Breast Neoplasms --drug therapy--DT; HL-60 Cells--drug effects--DE; Humans; Mice; Mice, SCID ; Neoplasm Transplantation; Oxidation-Reduction--drug effects--DE; Thioredoxin Reductase (NADPH)--drug effects--DE; Thioredoxin Reductase (NADPH)--metabolism--ME; Transplantation, Heterologous
CAS Registry No.: 0 (Antineoplastic Agents); 0 (Disulfides); 52500-60-4 (Thioredoxin)
Enzyme No.: EC 1.6.4.5 (Thioredoxin Reductase (NADPH))
Record Date Created: 19980112
Record Date Completed: 19980112

3/9/4
DIALOG(R)File 155:MEDLINE(R)

11979036 PMID: 9264384
Interleukin-15 + thioredoxin induce DNA synthesis in B-chronic lymphocytic leukemia cells but not in normal B cells.
Soderberg O; Christiansen I; Nilsson G; Carlsson M; Nilsson K
Department of Pathology, University of Uppsala, University Hospital, Sweden.
Leukemia - official journal of the Leukemia Society of America, Leukemia Research Fund, U.K (ENGLAND) Aug 1997, 11 (8) p1298-304, ISSN 0887-6924 Journal Code: 8704895
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
We have previously shown that Staphylococcus aureus Cowan strain 1 particles (SAC) + thioredoxin (Trx) + IL-2 may induce B-chronic lymphocytic leukemia (B-CLL) cells to proliferate. In this paper we have examined IL-15, which has activities similar to IL-2, for its ability to stimulate B-CLL cells and compared its activity with that of IL-2. We found that B-CLL cells could be induced to DNA synthesis upon treatment with IL-15 + Trx. The presence of Trx was essential for the IL-15-induced DNA synthesis. This contrasts to the effect of IL-15 + Trx on normal CD5+ and CD5- B cells, where IL-15 + Trx alone only induced limited DNA synthesis. IL-15 was as effective in the induction of DNA synthesis in B-CLL cells as IL-2, but about 100-fold less potent with an EC50 of 200 ng/ml. In addition we found that the IL-15 + Trx-induced proliferation was inhibited by CD40 stimulation. We conclude that IL-15 together with a proper costimulus can induce B-CLL cells to proliferate in vitro.
Tags: Research Support, Non-U.S. Gov't
Descriptors: *B-Lymphocytes--cytology--CY; *DNA--biosynthesis--BI; *Interleukin-15--administration and dosage--AD; *Leukemia, B-Cell, Chronic --pathology--PA; * **Thioredoxin** --administration and dosage--AD; Antigens, CD40--physiology--PH; Cell Cycle--drug effects--DE; Cell Differentiation --drug effects--DE; Cell Division--drug effects--DE; Humans; Lymphocyte Activation; Receptors, Interleukin-2--metabolism--ME; Tumor Cells, Cultured
CAS Registry No.: 0 (Antigens, CD40); 0 (Interleukin-15); 0 (Receptors, Interleukin-2); 52500-60-4 (Thioredoxin); 9007-49-2 (DNA)
Record Date Created: 19970904

Record Date Completed: 19970904

3/9/5
DIALOG(R)File 155:MEDLINE(R)

11766698 PMID: 9011676

Amelioration of ischemia-reperfusion injury by human thioredoxin in rabbit lung.

Okubo K; Kosaka S; Isowa N; Hirata T; Hitomi S; Yodoi J; Nakano M; Wada H
Department of Thoracic Surgery, Chest Disease Research Institute, Kyoto University, Japan.

Journal of thoracic and cardiovascular surgery (UNITED STATES) Jan 1997, 113 (1) p1-9, ISSN 0022-5223 Journal Code: 0376343
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: AIM; INDEX MEDICUS

Human thioredoxin is a polypeptide with thiol groups, possessing reducing activity, which is proved to have the ability to reduce active oxygens. This study evaluated the effect of human thioredoxin on the ischemia-reperfusion lung injury and the roles of human thioredoxin on active oxygens by chemiluminescence examination. The left hilum of the lung of Japanese white rabbits was occluded for 110 minutes and then reperfused for 90 minutes. Ten, 30, 60, and 90 minutes after reperfusion the right hilum was occluded for 5 minutes and the pulmonary functions of the left lung were examined. The animals were divided into four groups, three ischemia groups and a sham group (without occlusion; n = 6). The ischemia groups received human thioredoxin, 60 mg/kg (n = 10), N-acetylcysteine, 150 mg/kg (n = 7), or saline solution (control, n = 10) during reperfusion. Three rabbits in the human thioredoxin group and the control group were used to measure active oxygens with a cypridina luciferin analog. An additional group of reperfused lungs (n = 3) that were given superoxide dismutase after 110 minutes of ischemia was established to identify chemiluminescence examination. Compared with the sham group, reperfusion after 110 minutes of ischemia produced a significant lung injury in the control group. Among the ischemia groups, the human thioredoxin group showed significantly higher arterial oxygen tension at 30, 60, and 90 minutes after reperfusion than the control group, although there was no significant difference between the N-acetylcysteine and control groups. Histologically, intraalveolar exudation, interstitial thickening, and cellular infiltration were seen in the control group, whereas in the thioredoxin group alveolar structure was well preserved. In the measurement of active oxygens the chemiluminescence in the human thioredoxin group was less than that in the control group and as little as that in the group administered superoxide dismutase. We concluded human thioredoxin attenuated ischemia-reperfusion injury by involving active oxygens in rabbit lungs.

Tags: Male
Descriptors: *Lung--blood supply--BS; *Reperfusion Injury--drug therapy --DT; * **Thioredoxin** --therapeutic use--TU; Acetylcysteine--therapeutic use --TU; Animals; Chemiluminescent Measurements; Humans; Lung--pathology--PA; Oxygen--metabolism--ME; Rabbits; Reperfusion Injury--pathology--PA
CAS Registry No.: 52500-60-4 (Thioredoxin); 616-91-1 (Acetylcysteine); 7782-44-7 (Oxygen)
Record Date Created: 19970204

Record Date Completed: 19970204

3/9/6

DIALOG(R)File 155:MEDLINE(R)

11210808 PMID: 8545467

Proline, ascorbic acid, or thioredoxin affect jaundice and mortality in Long Evans cinnamon rats.

Hawkins R L; Mori M; Inoue M; Torii K

Torii Nutrient-stasis Project, Research Development Corporation of Japan, Yokohama, Japan.

Pharmacology, biochemistry, and behavior (UNITED STATES) Nov 1995, 52 (3) p509-15, ISSN 0091-3057 Journal Code: 0367050

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

The Long Evans Cinnamon (LEC) rat spontaneously develops fulminant hepatitis, which is usually lethal due to excess copper accumulation in the liver and is considered an animal model of Wilson's disease. LEC rats show a strong appetite for proline solution. Daily oral (p.o.) administration of proline resulted in significant delay of mortality. Feeding a copper-deficient diet greatly delayed the onset of jaundice and mortality and voluntary consumption or p.o. administration of proline further delayed jaundice and prevented mortality. LEC rats also consume ascorbic acid solutions, and p.o. administration of ascorbate also results in a significant delay in the appearance of jaundice and mortality. Combined treatment with ascorbic acid and proline is additive to delay further jaundice and mortality. An endogenous antioxidant protein, thioredoxin, when infused by minipump IP, could also inhibit the incidence of jaundice. These results indicate that antioxidant treatment combined with proline may be of benefit in Wilson's disease and possibly other forms of hepatic dysfunction.

Tags: Male

Descriptors: *Antioxidants--therapeutic use--TU; *Ascorbic Acid --therapeutic use--TU; *Jaundice--drug therapy--DT; *Proline--therapeutic use--TU; * **Thioredoxin** --therapeutic use--TU; Aging--physiology--PH; Animals; Antioxidants--administration and dosage--AD; Ascorbic Acid --administration and dosage--AD; Copper--deficiency--DF; Diet; Infusion Pumps, Implantable; Jaundice--genetics--GE; Jaundice--mortality--MO; Proline--administration and dosage--AD; Rats; Rats, Inbred Strains; **Thioredoxin** --administration and dosage--AD

CAS Registry No.: 0 (Antioxidants); 147-85-3 (Proline); 50-81-7 (Ascorbic Acid); 52500-60-4 (Thioredoxin); 7440-50-8 (Copper)

Record Date Created: 19960214

Record Date Completed: 19960214

3/9/7

DIALOG(R)File 155:MEDLINE(R)

11189281 PMID: 7577807

Suppression of adult T cell leukemia-derived factor/human thioredoxin induction by FK506 and cyclosporin A: a new mechanism of immune modulation

via redox control.

Furuke K; Nakamura H; Hori T; Iwata S; Maekawa N; Inamoto T; Yamaoka Y; Yodoi J

Institute for Virus Research, Kyoto University, Japan.

International immunology (ENGLAND) Jun 1995, '7 (6) p985-93, ISSN 0953-8178 Journal Code: 8916182

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Adult T cell leukemia-derived factor (ADF), which is identical to a disulfide reducing enzyme human thioredoxin (TRX), is produced and released by activated or virus-infected lymphocytes. Here we report that, in peripheral blood mononuclear cells (PBMC) stimulated with phytohemagglutinin (PHA), ADF/TRX mRNA was induced within 8 h after stimulation as detected by in situ hybridization study. To analyze the mechanism of ADF/TRX induction during T cell activation, the effects of immunosuppressants including FK506, rapamycin (Rap) and cyclosporin A (CsA) on ADF/TRX expression were investigated by immunoblot analysis. ADF/TRX induction in PBMC by PHA, Con A or OKT3 mAb was almost completely suppressed by FK506. Whereas CsA also inhibited ADF/TRX expression in OKT3 mAb-stimulated PBMC, Rap failed to affect it in spite of exhibiting growth inhibition. In addition, exogenous IL-2 could not increase ADF/TRX production in FK506-treated PBMC or in PHA blasts. These results indicate that ADF/TRX induction in T cell activation depends on calcineurin-dependent events in the early phase and that IL-2 production is not directly involved in ADF/TRX induction. Furthermore, when recombinant ADF (rADF) was added to a culture of PBMC 1 h before the addition of PHA and FK506, the action of FK506 was partially reversed as determined by [3H]thymidine incorporation and viable cell counts. These results suggest that ADF/TRX produced and released from PBMC may be a crucial event in lymphocyte activation, and that FK506 and CsA may exert the immune suppression partly through inhibiting the induction of the endogenous reducing factor ADF/TRX.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Adjuvants, Immunologic--pharmacology--PD; *Cyclosporine --pharmacology--PD; *Cytokines--drug effects--DE; *Neoplasm Proteins--drug effects--DE; *Tacrolimus--pharmacology--PD; * **Thioredoxin** --drug effects --DE; Cytokines--biosynthesis--BI; Cytokines--genetics--GE; Growth Inhibitors--pharmacology--PD; Humans; Immunosuppressive Agents --pharmacology--PD; Interleukin-2--pharmacology--PD; Leukocytes, Mononuclear--drug effects--DE; Neoplasm Proteins--biosynthesis--BI; Neoplasm Proteins--genetics--GE; Oxidation-Reduction--drug effects--DE; Polyenes --pharmacology--PD; RNA, Messenger--analysis--AN; Recombinant Proteins --pharmacology--PD; Sirolimus; Thioredoxin--biosynthesis--BI; Thioredoxin --genetics--GE

CAS Registry No.: 0 (Adjuvants, Immunologic); 0 (Cytokines); 0 (Growth Inhibitors); 0 (Immunosuppressive Agents); 0 (Interleukin-2); 0 (Neoplasm Proteins); 0 (Polyenes); 0 (RNA, Messenger); 0 (Recombinant Proteins); 0 (adult T cell leukemia-derived factor); 109581-93-3 (Tacrolimus); 52500-60-4 (Thioredoxin); 53123-88-9 (Sirolimus); 59865-13-3 (Cyclosporine)

Record Date Created: 19951212

Record Date Completed: 19951212

11137700 PMID: 7592733

Purification and functional analysis of the Mycobacterium leprae thioredoxin/thioredoxin reductase hybrid protein.

Wieles B; van Noort J; Drijfhout J W; Offringa R; Holmgren A; Ottenhoff T H

Department of Immunohematology, Leiden University Hospital, The Netherlands.

Journal of biological chemistry (UNITED STATES) Oct 27 1995, 270 (43) p25604-6, ISSN 0021-9258 Journal Code: 2985121R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS

In Mycobacterium leprae, thioredoxin and thioredoxin reductase are expressed from a single gene. This results in the expression of a hybrid protein with subunits attached to each other by a hydrophilic peptide linker. In all other organisms studied so far, thioredoxin (Trx) and thioredoxin reductase (TR) are expressed as two separate proteins. This raises the question of whether the hybrid protein is enzymatically active and, if so, whether TR reduces its own Trx partner or alternatively a heterologous Trx subunit. To address this question, the hybrid TR/Trx protein of M. leprae as well as the individual parts of the hybrid gene coding for either TR or Trx were overexpressed in Escherichia coli and purified. The purified proteins were tested for their ability to catalyze NADPH-dependent insulin disulfide reduction. Here we show that the M. leprae hybrid protein is indeed enzymatically active. Compared with the enzymatic activity of the separately expressed Trx and TR proteins, the hybrid protein is shown to be more efficient, particularly at low equimolar concentrations. This suggests that the hybrid protein of M. leprae is active by itself and that its activity involves intramolecular interactions between the TR and Trx domains. The activity of the hybrid protein increases when exogenous TR or Trx is added, indicating an additional role for intermolecular interactions.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Bacterial Proteins--metabolism--ME; *Mycobacterium leprae --enzymology--EN; *Thioredoxin--metabolism--ME; *Thioredoxin Reductase (NADPH)--metabolism--ME; Bacterial Proteins--genetics--GE; Bacterial Proteins--isolation and purification--IP; Disulfides--metabolism--ME; Escherichia coli--genetics--GE; Insulin--metabolism--ME; NADP--metabolism --ME; Recombinant Proteins--isolation and purification--IP; Recombinant Proteins--metabolism--ME; **Thioredoxin** --drug effects--DE; Thioredoxin --genetics--GE; Thioredoxin--isolation and purification--IP; Thioredoxin --pharmacology--PD; Thioredoxin Reductase (NADPH)--drug effects--DE; Thioredoxin Reductase (NADPH)--genetics--GE; Thioredoxin Reductase (NADPH)--isolation and purification--IP

CAS Registry No.: 0 (Bacterial Proteins); 0 (Disulfides); 0 (Recombinant Proteins); 0 (thioredoxin-thioredoxin reductase hybrid protein, Mycobacterium leprae); 11061-68-0 (Insulin); 52500-60-4 (Thioredoxin); 53-59-8 (NADP)

Enzyme No.: EC 1.6.4.5 (Thioredoxin Reductase (NADPH))

Record Date Created: 19951214
Record Date Completed: 19951214

3/9/9

DIALOG(R)File 155:MEDLINE(R)

11005228 PMID: 7785012

Attenuation of ischaemia reperfusion injury by human thioredoxin.

Fukuse T; Hirata T; Yokomise H; Hasegawa S; Inui K; Mitsui A; Hirakawa T; Hitomi S; Yodoi J; Wada H

Department of Thoracic Surgery, Kyoto University, Japan.

Thorax (ENGLAND) Apr 1995, 50 (4) p387-91, ISSN 0040-6376

Journal Code: 0417353

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

BACKGROUND--Active oxygen species are thought to play a part in ischaemia reperfusion injury. The ability of a novel agent, human thioredoxin (hTRX), to attenuate lung damage has been examined in a rat model of ischaemia reperfusion injury. METHODS--Twenty eight animals were studied. At thoracotomy the left main bronchus and the left main pulmonary artery were clamped for 75 minutes and the lung was then reperfused for 20 minutes. Phosphate buffered saline was administered intravenously to nine control animals and hTRX (30 micrograms/g body weight) was given intravenously to another group of nine animals. Two experiments were carried out. The first (Exp 1) was a time matched pair experiment (five treated, five controls), and the second (Exp 2) was performed under controlled conditions (four treated, four controls; temperature 25 degrees C, humidity 65%). In another 10 nonischaemic rats and those in Exp 1 biochemical measurements of lipid peroxide, superoxide dismutase, and glutathione peroxide levels were performed. RESULTS--In both experiments rats perfused with hTRX survived longer than controls. In Exp 1 the arterial oxygen tension (PaO2) on air in the hTRX group was higher at 20 minutes than at one minute after reperfusion. In Exp 2 PaO2 at 20 minutes was higher in the hTRX group than in the controls. Lipid peroxide, superoxide dismutase, and glutathione peroxide levels in the control group were higher than in the hTRX group and in the non-ischaemic groups. Histological examination showed less thickening and oedema of the alveolar walls in the hTRX group than in controls. CONCLUSIONS--These results suggest that hTRX is effective as a radical scavenger and can limit the extent of ischaemia reperfusion injury of the lungs of experimental animals.

Tags: Male

Descriptors: *Free Radical Scavengers--therapeutic use--TU; *Lung--drug effects--DE; *Reperfusion Injury--drug therapy--DT; * **Thioredoxin** --therapeutic use--TU; Animals; Glutathione Peroxidase--analysis--AN; Humans; Lipid Peroxides--analysis--AN; Lung--chemistry--CH; Oxygen--blood --BL; Rats; Rats, Wistar; Reperfusion Injury--blood--BL; Superoxide Dismutase--analysis--AN

CAS Registry No.: 0 (Free Radical Scavengers); 0 (Lipid Peroxides); 52500-60-4 (Thioredoxin); 7782-44-7 (Oxygen)

Enzyme No.: EC 1.11.1.9 (Glutathione Peroxidase); EC 1.15.1.1 (Superoxide Dismutase)

Record Date Created: 19950714

Record Date Completed: 19950714

3/9/10

DIALOG(R)File 155:MEDLINE(R)

10776571 PMID: 7979362

Thioredoxin: a multifunctional regulatory protein with a bright future in technology and medicine.

Buchanan B B; Schurmann P; Decottignies P; Lozano R M

Department of Plant Biology, University of California, Berkeley 94720.

Archives of biochemistry and biophysics (UNITED STATES) Nov 1 1994, 314 (2) p257-60, ISSN 0003-9861 Journal Code: 0372430

Publishing Model Print

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Thioredoxins are proteins, typically with a molecular mass of 12 kDa, that are widely, if not universally, distributed in the animal, plant, and bacterial kingdoms. Thioredoxins undergo reversible redox change through a disulfide group (S-S-->2 SH). Two cellular reductants--reduced ferredoxin and NADPH--supply the equivalents for reduction via different enzymes. The nature of the reductant serves as a basis for distinguishing and naming the two thioredoxin systems, which are discussed below in relation to their possible application in technology and medicine. Most of the discussion is referenced by general reviews. In the section dealing with animal cells, however, much of the material is quite recent. Thus, there, and elsewhere to a lesser extent, previously uncited studies are assigned specific references. (23 Refs.)

Descriptors: *Thioredoxin--metabolism--ME; * **Thioredoxin** --therapeutic use--TU; Animals; Embryonic and Fetal Development; Ferredoxins--metabolism --ME; Humans; NADP--metabolism--ME; Plants--cytology--CY; Plants --metabolism--ME

CAS Registry No.: 0 (Ferredoxins); 52500-60-4 (Thioredoxin); 53-59-8 (NADP)

Record Date Created: 19941207

Record Date Completed: 19941207

3/9/11

10197656 PMID: 1339022

Differences in hydrogen exchange behavior between the oxidized and reduced forms of Escherichia coli thioredoxin.

Kaminsky S M; Richards F M

Department of Molecular Biophysics and Biochemistry, Yale University, New Haven, Conneticut 06511.

Protein science - a publication of the Protein Society (UNITED STATES) Jan 1992, 1 (1) p10-21, ISSN 0961-8368 Journal Code: 9211750

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Amide proton exchange of thioredoxin is used to monitor the structural effects of reduction of its single disulfide. An effective 3-5-proton difference between the oxidized and reduced protein form is observed early in proton out-exchange of the whole protein, which is independent of

temperature in the range of 5-45 degrees C, indicating that redox-sensitive changes are probably not due to low-energy structural fluctuations. Medium resolution hydrogen exchange experiments have localized the redox-sensitive amide protons to two parts of the sequence that are distant from each other in the three-dimensional structure: the active-site turn and the first beta-strand. The sum of the proton differences observed in the peptides from these regions is equal to that of the whole protein, indicating that all redox-sensitive hydrogen exchange effects are observed in the peptide experiments. A model combining structural changes within the protein matrix with changes in the surface hydration properties is proposed as a mechanism for the communication between distant sites within the protein. Sound velocity and density measurements of reduced and oxidized thioredoxin are presented in the accompanying paper (Kaminsky, S.M. & Richards, F.M., 1992, Protein Sci. 1, 22-30).

Tags: Comparative Study

Descriptors: *Escherichia coli--enzymology--EN; *Thioredoxin--chemistry --CH; Amides--chemistry--CH; Models, Molecular; Oxidation-Reduction; Pepsin A--pharmacology--PD; Peptide Fragments--chemistry--CH; Protons; Signal Transduction; **Thioredoxin** --drug effects--DE; Tritium--chemistry--CH

CAS Registry No.: 0 (Amides); 0 (Peptide Fragments); 0 (Protons); 10028-17-8 (Tritium); 52500-60-4 (Thioredoxin)

Enzyme No.: EC 3.4.23.1 (Pepsin A)

Record Date Created: 19930713

Record Date Completed: 19930713

3/9/12

10163173 PMID: 8484715

Activation of oxidized cysteine proteinases by thioredoxin-mediated reduction in vitro.

Stephen A G; Powls R; Beynon R J

Department of Biochemistry, University of Liverpool, U.K.

Biochemical journal (ENGLAND) Apr 15 1993, 291 (Pt 2) p345-7, ISSN 0264-6021 Journal Code: 2984726R

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Activity of the cysteine adducts of the cysteine proteinases papain and thaumatopain can be recovered by treatment with thioredoxin, thioredoxin reductase and NADPH. Recovery of proteinase activity did not occur if any of the components of the thioredoxin system were omitted, or if thioredoxin or thioredoxin reductase were heat-inactivated. Such an enzyme-mediated process may be of significance in the recovery of cysteine proteinases inactivated by oxidative attack.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Cysteine Endopeptidases--metabolism--ME; *Thioredoxin --pharmacology--PD; Enzyme Activation--drug effects--DE; Kinetics; Oxidation-Reduction; Papain--metabolism--ME; Plants--enzymology--EN; **Thioredoxin** --administration and dosage--AD

CAS Registry No.: 52500-60-4 (Thioredoxin)

Enzyme No.: EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.22.- (thaumatopain); EC 3.4.22.2 (Papain)

Record Date Created: 19930601

Record Date Completed: 19930601

3/9/13

DIALOG(R)File 155:MEDLINE(R)

09695326 PMID: 1543487

Thioredoxin gene expression is transcriptionally up-regulated by retinol in monkey conducting airway epithelial cells.

An G; Wu R

California Primate Research Center, University of California, Davis 95616.

Biochemical and biophysical research communications (UNITED STATES) Feb 28 1992, 183 (1) p170-5, ISSN 0006-291X Journal Code: 0372516

Contract/Grant No.: ES00628; ES; NIEHS; HL35635; HL; NHLBI

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Using the differential hybridization technique, a cDNA clone, MT78, was isolated from the cDNA library of retinol-treated monkey tracheobronchial (TBE) epithelial cells. MT78 has a high sequence homology to human thioredoxin. The cDNA insert contains 506 nucleotides which encodes a peptide of 105 amino acids. The deduced peptide contains the highly conserved sequence Cys-Gly-Pro-Cys, found at the active site of all thioredoxins. The expression of the thioredoxin gene is stimulated 8-10 fold by vitamin A (retinol) in monkey TBE cells. The expression is significantly enhanced within 4 h after the vitamin A treatment and concurrent protein synthesis is not required for this enhancement. These results, in conjunction with the nuclear run-on transcriptional assay, support the conclusion that thioredoxin gene is transcriptionally up-regulated by retinol and/or its metabolites.

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Bronchi--drug effects--DE; *Gene Expression Regulation, Enzymologic--drug effects--DE; *Thioredoxin--genetics--GE; *Trachea--drug effects--DE; *Vitamin A--pharmacology--PD; Amino Acid Sequence; Animals; Base Sequence; Bronchi--cytology--CY; Cells, Cultured; Cloning, Molecular; Epithelial Cells; Epithelium--drug effects--DE; Macaca mulatta; Molecular Sequence Data; Restriction Mapping; **Thioredoxin** --drug effects--DE; Trachea--cytology--CY; Up-Regulation--drug effects--DE

Molecular Sequence Databank No.: GENBANK/M84643

CAS Registry No.: 11103-57-4 (Vitamin A); 52500-60-4 (Thioredoxin)

Record Date Created: 19920403

Record Date Completed: 19920403

3/9/14

DIALOG(R)File 155:MEDLINE(R)

08496400 PMID: 2492995

Thioredoxin is essential for photosynthetic growth. The thioredoxin m gene of Anacystis nidulans.

Muller E G; Buchanan B B

Division of Molecular Plant Biology, University of California, Berkeley

94720.
Journal of biological chemistry (UNITED STATES) Mar 5 1989, 264 (7) p4008-14, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant No.: 1 V41 RR-01685; RR; NCRR
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS

We have taken advantage of the transformation properties of the cyanobacterium Anacystis nidulans R2 to investigate the importance of thioredoxin for photosynthetic growth. The gene encoding thioredoxin m, designated trxM, was cloned from A. nidulans using a synthetic oligonucleotide probe. Based on the nucleotide sequence, thioredoxin m of A. nidulans is composed of 107 amino acids and shares 84, 48, and 48% sequence identity with thioredoxins from Anabaena, spinach, and Escherichia coli, respectively. The trxM gene is single copy and is transcribed on a 510-nucleotide mRNA. We demonstrate that disruption of the trxM gene with a kanamycin resistance gene cartridge is a lethal mutation. Although dispensable in E. coli, thioredoxin is essential for the photosynthetic growth of A. nidulans.

Tags: Research Support, U.S. Gov't, Non-P.H.S.; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Bacterial Proteins--physiology--PH; *Cyanobacteria --physiology--PH; *Photosynthesis; * **Thioredoxin** --physiology--PH; Amino Acid Sequence; Base Sequence; Cloning, Molecular; DNA Mutational Analysis; Escherichia coli--genetics--GE; Gene Expression Regulation; Genes, Lethal; Genes, Structural; Molecular Sequence Data; Oligonucleotide Probes; Plasmids; Restriction Mapping; Transformation, Genetic

Molecular Sequence Databank No.: GENBANK/J04475
CAS Registry No.: 0 (Bacterial Proteins); 0 (Oligonucleotide Probes); 0 (Plasmids); 52500-60-4 (Thioredoxin)
Record Date Created: 19890403
Record Date Completed: 19890403

3/9/15
DIALOG(R)File 155:MEDLINE(R)

08337479 PMID: 3044397
Bacterial and mammalian thioredoxin systems activate iodothyronine 5'-deiodination.
Das A K; Hummel B C; Gleason F K; Holmgren A; Walfish P G
Thyroid Research Laboratory, Mount Sinai Hospital, Toronto, Ont., Canada.
Biochemistry and cell biology = Biochimie et biologie cellulaire (CANADA) May 1988, 66 (5) p460-4, ISSN 0829-8211 Journal Code: 8606068
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS

The identity of a dithiol (designated DFB) of relative mass (Mr) = 13,000, reported previously to be present in fraction B of rat liver cytosol and to participate as a cofactor in the 5'-deiodination of iodothyronines, has been investigated. Substitution of highly purified thioredoxin from Escherichia coli for fraction B or of highly purified

thioredoxin reductase from either E. coli or rat liver for cytosolic fraction A (containing DFB reductase) permits deiodination of 3,3',5'-[125I]triiodothyronine by rat liver microsomes to proceed. Addition of antibodies to highly purified rat-liver thioredoxin or thioredoxin reductase inhibits deiodination. Thus, the thioredoxin system largely accounts for the activity of the cytosolic cofactor system supporting 5'-deiodination of 3,3',5'-triiodothyronine in rat liver.

Descriptors: *Bacterial Proteins--physiology--PH; *NADH, NADPH Oxidoreductases--physiology--PH; * **Thioredoxin** --physiology--PH; *Thioredoxin Reductase (NADPH)--physiology--PH; *Thyronines--metabolism--ME ; Animals; Cattle; Enzyme Activation; Escherichia coli; Microsomes, Liver --metabolism--ME; Rats

CAS Registry No.: 0 (Bacterial Proteins); 0 (Thyronines); 4732-82-5 (3'-monoiodothyronine); 52500-60-4 (Thioredoxin)

Enzyme No.: EC 1.6. (NADH, NADPH Oxidoreductases); EC 1.6.4.5 (Thioredoxin Reductase (NADPH))

Record Date Created: 19881013

Record Date Completed: 19881013

3/9/16

DIALOG(R)File 155:MEDLINE(R)

08178136 PMID: 11536590 Record Identifier: 00006435

Contrasting modes of photosynthetic enzyme regulation in oxygenic and anoxygenic prokaryotes.

Crawford N A; Sutton C W; Yee B C; Johnson T C; Carlson D C; Buchanan B B

Division of Molecular Plant Biology, University of California, Berkeley 94720, USA.

Archives of microbiology (GERMANY) Oct 1984, 139 (2-3) p124-9, ISSN 0302-8933 Journal Code: 0410427

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NASA

Other Citation Owner: NASA

Record type: MEDLINE; Completed

Enzymes that are regulated by the ferredoxin/thioredoxin system in chloroplasts--fructose-1,6-bis-phosphatase (FBPase), sedoheptulose-1,7-bisp hosphatase (SBPase), and phosphoribulokinase (PRK)--were partially purified from two different types of photosynthetic prokaryotes (cyanobacteria, purple sulfur bacteria) and tested for a response to thioredoxins. Each of the enzymes from the cyanobacterium Nostoc muscorum, an oxygenic organism known to contain the ferredoxin/thioredoxin system, was activated by thioredoxins that had been reduced either chemically by dithiothreitol or photochemically by reduced ferredoxin and ferredoxin-thioredoxin reductase. Like their chloroplast counterparts, N. muscorum FBPase and SBPase were activated preferentially by reduced thioredoxin f. SBPase was also partially activated by thioredoxin m. PRK, which was present in two regulatory forms in N. muscorum, was activated similarly by thioredoxins f and m. Despite sharing the capacity for regulation by thioredoxins, the cyanobacterial FBPase and SBPase target enzymes differed antigenically from their chloroplast counterparts. The corresponding enzymes from Chromatium vinosum, an anoxygenic photosynthetic purple bacterium found recently to contain the NADP/thioredoxin system, differed from both those of cyanobacteria and chloroplasts in showing no response to reduced thioredoxin. Instead, C. vinosum FBPase, SBPase, and PRK activities were regulated by a metabolite effector, 5'-AMP. The evidence is in accord with

the conclusion that thioredoxins function in regulating the reductive pentose phosphate cycle in oxygenic prokaryotes (cyanobacteria) that contain the ferredoxin/thioredoxin system, but not in anoxygenic prokaryotes (photosynthetic purple bacteria) that contain the NADP/thioredoxin system. In organisms of the latter type, enzyme effectors seem to play a dominant role in regulating photosynthetic carbon dioxide assimilation.

Tags: Comparative Study; Research Support, U.S. Gov't, Non-P.H.S.

Descriptors: *Chromatium--enzymology--EN; *Cyanobacteria--enzymology--EN; *Ferredoxins--physiology--PH; *Photosynthesis--physiology--PH; *Thioredoxin --metabolism--ME; Adenosine Monophosphate--metabolism--ME; Adenosine Monophosphate--physiology--PH; Aerobiosis; Anaerobiosis; Dithiothreitol --pharmacology--PD; Enzyme Activation; Ferredoxins--metabolism--ME; Fructose-Bisphosphatase--metabolism--ME; NADP--metabolism--ME; NADP --physiology--PH; Oxidoreductases--metabolism--ME; Oxidoreductases --physiology--PH; Pentosephosphate Pathway--drug effects--DE; Pentosephosphate Pathway--physiology--PH; Phosphoric Monoester Hydrolases --metabolism--ME; Phosphotransferases (Alcohol Group Acceptor)--metabolism --ME; Sulfhydryl Reagents--pharmacology--PD; **Thioredoxin** --drug effects --DE

CAS Registry No.: 0 (Ferredoxins); 0 (Sulfhydryl Reagents); 3483-12-3 (Dithiothreitol); 52500-60-4 (Thioredoxin); 53-59-8 (NADP); 61-19-8 (Adenosine Monophosphate)

Enzyme No.: EC 1. (Oxidoreductases); EC 1.18.- (ferredoxin-thioredoxin reductase); EC 2.7.1 (Phosphotransferases (Alcohol Group Acceptor)); EC 2.7.1.19 (phosphoribulokinase); EC 3.1.3 (Phosphoric Monoester Hydrolases); EC 3.1.3.11 (Fructose-Bisphosphatase); EC 3.1.3.37 (sedoheptulose-bisphosphatase)

Record Date Created: 19980828

Record Date Completed: 19980828

3/9/17

08055903 PMID: 3682813

The heat-stable cytosolic factor that promotes glucocorticoid receptor binding to DNA is neither thioredoxin nor ribonuclease.

Tienrungroj W; Pratt S E; Grippo J F; Holmgren A; Pratt W B

Department of Pharmacology, University of Michigan Medical School, Ann Arbor 48109-0010.

Journal of steroid biochemistry (ENGLAND) Nov 1987, 28 (5) p449-57, ISSN 0022-4731 Journal Code: 0260125

Contract/Grant No.: AM31573; AM; NIADDK

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Treatment of rat liver cytosol containing temperature-transformed [3H]dexamethasone-bound receptors at 0 degree C with the sulfhydryl modifying reagent methyl methanethiosulfonate (MMTS) inhibits the DNA-binding activity of the receptor, and DNA-binding activity is restored after addition of dithiothreitol (DTT). However, transformed receptors that are treated with MMTS and then separated from low Mr components of cytosol by passage through a column of Sephadex G-50 have very little DNA-binding activity when DTT is added to regenerate sulfhydryl moities. The receptors

will bind to DNA if whole liver cytosol or boiled liver cytosol is added in addition to DTT. The effect of boiled cytosol is mimicked by purified rat thioredoxin or bovine RNase A in a manner that does not reflect the reducing activity of the former or the catalytic activity of the latter. This suggests that the reported ability of each of these heat-stable peptides to stimulate DNA binding by glucocorticoid receptors is not a biologically relevant action. We suggest that stimulation of DNA binding of partially purified receptors by boiled cytosol does not constitute a reconstitution of a complete cytosolic system in which the dissociated receptor must associate with a specific heat-stable accessory protein required for DNA binding, as has been suggested in the "two-step" model of receptor transformation recently proposed by Schmidt et al. (Schmidt T.J., Miller-Diener, A., Webb M.L. and Litwack G. (1985) J. biol. Chem. 260, 16255-16262).

Tags: Male; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Bacterial Proteins--physiology--PH; *Cytosol--physiology --PH; *DNA--metabolism--ME; *Liver--metabolism--ME; *Peptides--physiology --PH; *Receptors, Glucocorticoid--metabolism--ME; *Ribonucleases --physiology--PH; * **Thioredoxin** --physiology--PH; Animals; Dexamethasone --metabolism--ME; Dithiothreitol--pharmacology--PD; Heat; Kinetics; Methyl Methanesulfonate--analogs and derivatives--AA; Methyl Methanesulfonate --pharmacology--PD; Protein Binding; Rats; Rats, Inbred Strains

CAS Registry No.: 0 (Bacterial Proteins); 0 (Peptides); 0 (Receptors, Glucocorticoid); 2949-92-0 (methyl methanethiosulfonate); 3483-12-3 (Dithiothreitol); 50-02-2 (Dexamethasone); 52500-60-4 (Thioredoxin); 66-27-3 (Methyl Methanesulfonate); 9007-49-2 (DNA)

Enzyme No.: EC 3.1.- (Ribonucleases)

Record Date Created: 19871223

Record Date Completed: 19871223

3/9/18

DIALOG(R)File 155:MEDLINE(R)

07776037 PMID: 3492466

The effect of thioredoxin on the radiosensitivity of bacteria.

Lunn C A; Pigiet V P

International journal of radiation biology and related studies in physics, chemistry, and medicine (ENGLAND) Jan 1987, 51 (1) p29-38, ISSN 0020-7616 Journal Code: 0374725

Contract/Grant No.: 5T32-GM7231; GM; NIGMS; GM23823; GM; NIGMS

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

The ability of Escherichia coli thioredoxin to protect cells from lethal amounts of gamma radiation was tested using bacterial strains engineered to contain different amounts of thioredoxin per cell. Cells grown to late stationary phase demonstrated a decreasing sensitivity to gamma-radiation with increasing amounts of thioredoxin per cell. Exponentially growing cells were equally sensitive to the gamma-radiation regardless of the intracellular concentration of thioredoxin. Cells exhibiting the radiation-resistant phenotype in the stationary phase reverted to the radiation-sensitive phenotype when diluted into fresh growth medium. These results suggest that thioredoxin can protect cells from gamma-radiation

under certain metabolic conditions.
Tags: Research Support, U.S. Gov't, P.H.S.
Descriptors: *Bacteria--radiation effects--RE; *Bacterial Proteins --physiology--PH; * **Thioredoxin** --physiology--PH; Radiation Tolerance
CAS Registry No.: 0 (Bacterial Proteins); 52500-60-4 (Thioredoxin)
Record Date Created: 19870316
Record Date Completed: 19870316

3/9/19
DIALOG(R)File 155:MEDLINE(R)

07476826 PMID: 3004333 Record Identifier: 86129342
Enzyme regulation in C4 photosynthesis: purification, properties, and activities of thioredoxins from C4 and C3 plants.
Crawford N A; Yee B C; Hutcheson S W; Wolosiuk R A; Buchanan B B
Archives of biochemistry and biophysics (UNITED STATES) Jan 1986, 244 (1) p1-15, ISSN 0003-9861 Journal Code: 0372430
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Other Citation Owner: NASA
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
Procedures are described for the purification to homogeneity of chloroplast thioredoxins f and m from leaves of corn (Zea mays, a C4 plant) and spinach (Spinacea oleracea, a C3 plant). The C3 and C4f thioredoxins were similar immunologically and biochemically, but differed in certain of their physiochemical properties. The f thioredoxins from the two species were capable of activating both NADP-malate dehydrogenase (EC 1.1.1.37) and fructose-1,6-bisphosphatase (EC 3.1.3.11) when tested in standard thioredoxin assays. Relative to its spinach counterpart, corn thioredoxin f showed a greater molecular mass (15.0-16.0 kDa vs 10.5 kDa), lower isoelectric point (ca. 5.2 vs 6.0), and lower ability to form a stable noncovalent complex with its target fructose bisphosphatase enzyme. The C3 and C4 m thioredoxins were similar in their specificity (ability to activate NADP-malate dehydrogenase, and not fructose-1,6-bisphosphatase) and isoelectric points (ca. 4.8), but differed slightly in molecular mass (13.0 kDa for spinach vs 13.5 kDa for corn) and substantially in their immunological properties. Results obtained in conjunction with these studies demonstrated that the thioredoxin m-linked activation of NADP-malate dehydrogenase in selectively enhanced by the presence of halide ions (e.g., chloride) and by an organic solvent (e.g., 2-propanol). The results suggest that in vivo NADP-malate dehydrogenase interacts with thylakoid membranes and is regulated to a greater extent by thioredoxin m than thioredoxin f.
Tags: Research Support, U.S. Gov't, Non-P.H.S.
Descriptors: *Bacterial Proteins--isolation and purification--IP; *Malate Dehydrogenase--metabolism--ME; *Photosynthesis; *Plant Proteins--isolation and purification--IP; *Thioredoxin--isolation and purification--IP; Chloroplasts--metabolism--ME; Chromatography, Affinity; Electrophoresis, Polyacrylamide Gel; Enzyme Activation; Fructose-Bisphosphatase--metabolism --ME; Immunochemistry; Isoelectric Point; NADP--metabolism--ME; Plant Proteins--physiology--PH; Plants--metabolism--ME; Protein Binding; Salts --pharmacology--PD; Solvents--pharmacology--PD; **Thioredoxin** --physiology --PH; Zea mays--metabolism--ME
CAS Registry No.: 0 (Bacterial Proteins); 0 (Plant Proteins); 0

(Salts); 0 (Solvents); 0 (thioredoxin f); 52500-60-4 (Thioredoxin); 53-59-8 (NADP)
Enzyme No.: EC 1.1.1.37 (Malate Dehydrogenase); EC 3.1.3.11 (Fructose-Bisphosphatase)
Record Date Created: 19860312
Record Date Completed: 19860312

3/9/20

DIALOG(R)File 155:MEDLINE(R)

07320587 PMID: 3896121
Thioredoxin.
Holmgren A
Annual review of biochemistry (UNITED STATES) 1985, 54 p237-71, ISSN 0066-4154 Journal Code: 2985150R
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
(202 Refs.)
Tags: Comparative Study; Research Support, Non-U.S. Gov't
Descriptors: *Bacterial Proteins--physiology--PH; * **Thioredoxin** --physiology--PH; Animals; Bacteriophages--physiology--PH; Cell Compartmentation; Disulfides; Escherichia coli--physiology--PH; Evolution; Humans; Oxidoreductases--metabolism--ME; Plants; Ribonucleotide Reductases --metabolism--ME; Structure-Activity Relationship; Thioredoxin Reductase (NADPH)--metabolism--ME
CAS Registry No.: 0 (Bacterial Proteins); 0 (Disulfides); 52500-60-4 (Thioredoxin)
Enzyme No.: EC 1. (Oxidoreductases); EC 1.17.4 (Ribonucleotide Reductases); EC 1.6.4.5 (Thioredoxin Reductase (NADPH))
Record Date Created: 19850904
Record Date Completed: 19850904

3/9/21

DIALOG(R)File 155:MEDLINE(R)

07158227 PMID: 3881756
Thioredoxin is required for filamentous phage assembly.
Russel M; Model P
Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Jan 1985, 82 (1) p29-33, ISSN 0027-8424 Journal Code: 7505876
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
Sequence comparisons show that the fip gene product of Escherichia coli, which is required for filamentous phage assembly, is thioredoxin. Thioredoxin serves as a cofactor for reductive processes in many cell types and is a constituent of phage T7 DNA polymerase. The fip-1 mutation makes

filamentous phage and T7 growth temperature sensitive in cells that carry it. The lesion lies within a highly conserved thioredoxin active site. Thioredoxin reductase (NADPH), as well as thioredoxin, is required for efficient filamentous phage production. Mutant phages defective in phage gene I are particularly sensitive to perturbations in the fip-thioredoxin system. A speculative model is presented in which thioredoxin reductase, thioredoxin, and the gene I protein interact to drive an engine for filamentous phage assembly.

Tags: Research Support, U.S. Gov't, Non-P.H.S.; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Bacterial Proteins--physiology--PH; *Coliphages--growth and development--GD; * **Thioredoxin** --physiology--PH; *Virus Replication; Amino Acid Sequence; Base Sequence; Escherichia coli--genetics--GE; Genes, Bacterial; Morphogenesis; Mutation; Thioredoxin--genetics--GE

CAS Registry No.: 0 (Bacterial Proteins); 52500-60-4 (Thioredoxin)

Record Date Created: 19850227

Record Date Completed: 19850227

3/9/22

DIALOG(R)File 155:MEDLINE(R)

06565618 PMID: 6299735

The use of affinity chromatography on 2'5' ADP-sepharose reveals a requirement for NADPH, thioredoxin and thioredoxin reductase for the maintenance of high protein synthesis activity in rabbit reticulocyte lysates.

Hunt T; Herbert P; Campbell E A; Delidakis C; Jackson R J

European journal of biochemistry / FEBS (GERMANY, WEST) Mar 15 1983, 131 (2) p303-11, ISSN 0014-2956 Journal Code: 0107600

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Bacterial Proteins--physiology--PH; *Blood Proteins --biosynthesis--BI; *NADH, NADPH Oxidoreductases--physiology--PH; *NADP --physiology--PH; *Reticulocytes--metabolism--ME; * **Thioredoxin** --physiology--PH; *Thioredoxin Reductase (NADPH)--physiology--PH; Animals; Chromatography, Affinity; Cyclic AMP--pharmacology--PD; Glutathione --pharmacology--PD; Guanosine Triphosphate--pharmacology--PD; Rabbits; Sepharose--analogs and derivatives--AA; Sugar Phosphates--physiology--PH

CAS Registry No.: 0 (2',5'-ADP-sepharose); 0 (Bacterial Proteins); 0 (Blood Proteins); 0 (Sugar Phosphates); 52500-60-4 (Thioredoxin); 53-59-8 (NADP); 60-92-4 (Cyclic AMP); 70-18-8 (Glutathione); 86-01-1 (Guanosine Triphosphate); 9012-36-6 (Sepharose)

Enzyme No.: EC 1.6. (NADH, NADPH Oxidoreductases); EC 1.6.4.5 (Thioredoxin Reductase (NADPH))

Record Date Created: 19830527

Record Date Completed: 19830527

3/9/23

DIALOG(R)File 155:MEDLINE(R)

06565617 PMID: 6832153

The preparation and properties of gel-filtered rabbit-reticulocyte lysate protein-synthesis systems.

Jackson R J; Campbell E A; Herbert P; Hunt T

European journal of biochemistry / FEBS (GERMANY, WEST) Mar 15 1983, 131 (2) p289-301, ISSN 0014-2956 Journal Code: 0107600

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Tags: Female; Male; Research Support, Non-U.S. Gov't

Descriptors: *Blood Proteins--biosynthesis--BI; *Reticulocytes --metabolism--ME; Adenosine Triphosphate--pharmacology--PD; Animals; Cell-Free System; Chemistry; Chromatography, Gel; Glucose-6-Phosphate; Glucosephosphates--pharmacology--PD; Glutathione--pharmacology--PD; Guanosine Triphosphate--pharmacology--PD; Magnesium--pharmacology--PD; Oxidation-Reduction; Polyamines--pharmacology--PD; Rabbits; **Thioredoxin** --physiology --PH

CAS Registry No.: 0 (Blood Proteins); 0 (Glucosephosphates); 0 (Polyamines); 52500-60-4 (Thioredoxin); 56-65-5 (Adenosine Triphosphate); 56-73-5 (Glucose-6-Phosphate); 70-18-8 (Glutathione); 7439-95-4 (Magnesium); 86-01-1 (Guanosine Triphosphate)

Record Date Created: 19830527

Record Date Completed: 19830527

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22may05 13:31:55 User228206 Session D2447.2

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$8.63 Estimated cost this search

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L1: Entry 27 of 56 File: USPT Apr 9, 2002

US-PAT-NO: 6369294
DOCUMENT-IDENTIFIER: US 6369294 B1
** See image for Certificate of Correction **

TITLE: Methods comprising apoptosis inhibitors for the generation of transgenic pigs

DATE-ISSUED: April 9, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Piedrahita; Jorge A.	College Station	TX		
Bazer; Fuller W.	College Station	TX		

US-CL-CURRENT: 800/14; 435/325, 435/383, 435/384, 435/455, 435/459, 435/461, 435/462, 435/463, 800/17

CLAIMS:

What is claimed is:

1. A method of growing porcine primordial germ cells, comprising growing a cell culture comprising porcine primordial germ cells from an embryo of a pig on feeder cells for a time sufficient to obtain undifferentiated porcine primordial germ cells, said feeder cells at a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 cells/cm.sup.2, in a culture medium comprising an effective amount of basic fibroblast growth factor and an apoptosis inhibitor.

2. The method of claim 1, wherein said feeder cells are STO feeder cells.

3. The method of claim 1, wherein said culture medium comprises basic fibroblast growth factor at a concentration of between about 5 ng/ml and about 100 .mu.g/ml.

4. The method of claim 1, wherein said apoptosis inhibitor is a serine protease apoptosis inhibitor.

5. The method of claim 1, wherein said apoptosis inhibitor is an antioxidant apoptosis inhibitor.

6. The method of claim 1, wherein said apoptosis inhibitor is selected from the group consisting of bcl-2, Bcl-x1, Mcl-1, Bak, A1, A20, TNF-.alpha., nerve growth factor, epidermal growth factor, insulin-like growth factor-I, insulin-like growth factor-I receptor, 3 -aminobenzamide, aphidocolin, L-ascorbic acid, cataiase, follicle stimulating hormone, vasoactive intestinal peptide, cyclic GMP, hCG, interleukin-1.beta., superoxide dismutase, aurintricarboxylic acid, BAPTA/AM, caffeine, calpain inhibitor I, calpain inhibitor II, leupeptin, N-ethyl maleimaide, cyclosporin A, acetyl-leucyl-leucyl-

normethional, 3,4-dichloroisocoumarin, diisopropylfluorophosphate, phenylmethylsulfonyl fluoride, tetrodotoxin, nimodipine, verapamil, flunarizine, R56865, forskolin, genistein, herbimycin A, interleukin-6, calyculin A, okadaic acid, calyculin A, phorbol-12-myristate-13-acetate, 1-Pyrrolidinecarbodithioic acid, nifedipine, nisoldipine, aurintricarboxylic acid, spermine, thioredoxin, N.sup..alpha. -Tosyl-L-Phe chloromethyl ketone and N.sup..alpha. -Tosyl-L-Lys chloromethyl ketone.

7. The method of claim 6, wherein said apoptosis inhibitor is N-Acetyl-L-cysteine.

8. The method of claim 1, wherein said culture medium further comprises an effective amount of uteroferrin.

9. The method of claim 1, wherein said culture medium further comprises an effective amount of amino acids non-essential with respect to said pig.

10. The method of claim 1, wherein said culture medium further comprises an effective amount of L-glutamine.

11. The method of claim 1, wherein said culture medium further comprises an effective amount of P-mercaptoethanol.

12. The method of claim 1, wherein the porcine primordial germ cells are maintained in an undifferentiated state for between about 2 passages and about 14 passages.

13. The method of claim 1, wherein the porcine primordial germ cells comprise at least a first selected DNA segment.

14. The method of claim 13, wherein said selected DNA segment comprises at least a first coding region encoding a selected protein.

15. A method of preparing a porcine primordial germ cell-derived cell line, comprising:

(a) plating a cell culture comprising porcine primordial germ cells on feeder cells, said feeder cells at a density of between, about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 cells/cm.sup.2, in a culture medium comprising an effective amount of basic fibroblast growth factor and an apoptosis inhibitor; and

(b) culturing the plated porcine primordial germ cells for a period of time effective to provide a porcine primordial germ cell-derived cell line.

16. A method of preparing porcine primordial germ cells that contain a selected DNA segment, comprising:

(a) introducing said selected DNA segment into a composition comprising porcine primordial germ cells to obtain candidate porcine primordial germ cells that contain said selected DNA segment; and

(b) plating said candidate porcine primordial germ cells that contain said selected DNA segment on feeder cells, said feeder cells at a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 cells/cm.sup.2, in a culture medium comprising an effective amount of basic

fibroblast growth factor and an apoptosis inhibitor, to obtain said porcine primordial germ cells that contain said selected DNA segment.

17. The method of claim 16, wherein said apoptosis inhibitor is a serine protease apoptosis inhibitor.

18. The method of claim 16, wherein said apoptosis inhibitor is an antioxidant apoptosis inhibitor.

19. The method of claim 16, wherein said apoptosis inhibitor is selected from the group consisting of bcl-2, Bcl-x1, Mcl-1, Bak, A1, A20, TNF-.alpha., nerve growth factor, epidermal growth factor, insulin-like growth factor-I, insulin-like growth factor-I receptor, 3-aminobenzamide, aphidocolin, L-ascorbic acid, catalase, follicle stimulating hormone, vasoactive intestinal peptide, cyclic GMP, hCG, interleukin-1.beta., superoxide dismutase, aurintricarboxylic acid, BAPTA/AM, caffeine, calpain inhibitor I, calpain inhibitor II, leupeptin, N-ethyl maleimide, cyclosporin A, acetyl-leucyl-leucyl-normethional, 3,4-dichloroisocoumarin, diisopropylfluorophosphate, phenylmethylsulfonyl fluoride, tetrodotoxin, nimodipine, verapamil, flunarizine, R56865, forskolin, genistein, herbimycin A, interleukin-6, calyculin A, okadaic acid, calyculin A, phorbol-12-myristate-13-acetate, 1-Pyrrolidinecarbodithioic acid, nifedipine, nisoldipine, aurintricarboxylic acid, spermine, thioredoxin, N.sup..alpha. -Tosyl-L-Phe chloromethyl ketone and N.sup..alpha. -Tosyl-L-Lys chloromethyl ketone.

20. The method of claim 19, wherein said apoptosis inhibitor is N-Acetyl-L-cysteine.

21. The method of claim 16, further comprising culturing said porcine primordial germ cells that contain said selected DNA segment for between about 2 and about 14 passages.

22. The method of claim 16, wherein said selected DNA segment is introduced into said porcine primordial germ cells by electroporation, particle bombardment or viral transformation.

23. The method of claim 16, wherein said selected DNA segment comprises at least a first coding region encoding a selected protein.

24. The method of claim 23, wherein said first coding region encodes a selected marker protein.

25. The method of claim 23, wherein said first coding region encodes a protein, wherein said protein is an interleukin, collagen, interferon, blood protein, hormone, growth factor, cytokine, enzyme, receptor, binding protein, immune system protein, antigen, muscle protein or oncogene receptor.

26. The method of claim 25, wherein said first coding region encodes a protein, wherein said protein is a SREHP, GP63, actinobacillus, pleuropneumoniae, pseudomonas aeruynosa, OprF, myclin basic protein, insulin, hCD59, DAF (CD55), factor IX, urokinase, (x-antitrypsin, tissue plasminogen activator, protein C, activin, adenosine deaminase, angiotensinogen I, antithrombin III, alpha I antitrypsin, apolipoprotein A-I, apolipoprotein A-II, apolipoprotein C-I, apolipoprotein C-II, apolipoprotein C-III, apolipoprotein E, atrial natriuretic factor, chorionic gonadotropin, alpha chain, beta chain, pro (rennin) chymosin, factor B complement, complement C2, complement C3, complement C4, complement C9, corticotropin releasing factor,

epidermal growth factor, c-erb B, epoxide dehydratase, erythropoietin, C1 esterase inhibitor, factor VIII, factor IX, Christmas factor, factor X, fibrinogen A alpha, gamma B beta, gastrin releasing peptide, prepro glucagon, growth hormone, RF growth hormone, somatocrinin, hemopexin, inhibin, prepro insulin, insulin-like growth factor I, insulin-like growth factor II, alpha interferon, multiple leukocyte, fibroblast beta interferon, gamma interferon, interleukin-1, T-cell interleukin-2, growth factor, interleukin-3, two forms kininogen, beta subunit leuteinizing hormone, leuteinizing hormone, releasing hormone, lymphotoxin, mast cell growth factor, beta subunit nerve growth factor, PGDF c-sis oncogene, chain A, pancreatic polypeptide, icosapeptide, parathyroid hormone, prepro plasminogen, plasminogen activator, prolactin, proopiomelanocortin, protein C, prothrombin, relaxin, prepro renin, somatostatin, prepro tachykinin, substance P, substance K, urokinase or prepro vasoactive intestinal peptide protein.

27. The method of claim 23, wherein said selected DNA segment further comprises at least a second coding region encoding a selected protein.

28. The method of claim 27, wherein said first coding region encodes a selected protein and said second coding region encodes a selected marker protein.

29. The method of claim 16, wherein said DNA segment is operatively positioned under the control of a promoter.

30. The method of claim 16, wherein said selected DNA segment is operatively positioned in reverse orientation under the control of a promoter, wherein said promoter directs the expression of an antisense product.

31. The method of claim 16, wherein said DNA segment further comprises two selected DNA regions that flank said DNA segment, thereby directing the homologous recombination of said DNA segment into genomic DNA of said porcine primordial germ cells.

32. The method of claim 31, wherein said selected DNA regions comprise porcine genomic DNA.

33. The method of claim 31, wherein said DNA segment further comprises two selected DNA sequences that flank said DNA segment, thereby directing excision of said DNA segment under appropriate conditions.

34. A method of producing a transgenic pig comprising:

(a) introducing a selected DNA segment into a cell culture comprising porcine primordial germ cells to obtain candidate porcine primordial germ cells that contain said selected DNA segment;

(b) plating said candidate porcine primordial germ cells that contain said selected DNA segment on feeder cells, said feeder cells at a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 cells/m.sup.2, in a culture medium comprising an effective amount of basic fibroblast growth factor and an apoptosis inhibitor, to obtain undifferentiated porcine primordial germ cells that contain said selected DNA segment; and

(c) generating a transgenic pig from said undifferentiated porcine primordial germ cells that contain said selected DNA segment wherein said selected DNA

segment is contained and expressed in somatic and germ cells of said transgenic pig.

35. The method of claim 34, wherein said apoptosis inhibitor is a serine protease apoptosis inhibitor.

36. The method of claim 34, wherein said apoptosis inhibitor is an antioxidant apoptosis inhibitor.

37. The method of claim 34, wherein said apoptosis inhibitor is selected from the group consisting of bcl-2, Bcl-x1, Mcl-1, Bak, A1, A20, TNF-.alpha., nerve growth factor, epidermal growth factor, insulin-like growth factor-I, insulin-like growth factor-I receptor, 3-aminobenzamide, aphidocolin, L-ascorbic acid, catalase, follicle stimulating hormone, vasoactive intestinal peptide, cyclic GMP, hCG, interleukin-1.beta., superoxide dismutase, aurintricarboxylic acid, BAPTA/AM, caffeine, calpain inhibitor I, calpain inhibitor II, leupeptin, N-ethyl maleimide, cyclosporin A, acetyl-leucyl-leucyl-normethional, 3,4-dichloroisocoumarin, diisopropylfluorophosphate, phenylmethylsulfonyl fluoride, tetrodotoxin, nimodipine, verapamil, flunarizine, R56865, forskolin, genistein, herbimycin A, interleukin-6, calyculin A, okadaic acid, calyculin A, phorbol-12-myristate-13-acetate, 1-Pyrrolidinecarbodithioic acid, nifedipine, nisoldipine, aurintricarboxylic acid, spermine, thioredoxin, N.sup..alpha. -Tosyl-L-Phe chloromethyl ketone and N.sup..alpha. -Tosyl-L-Lys chloromethyl ketone.

38. The method of claim 37, wherein said apoptosis inhibitor is N-Acetyl-L-cysteine.

39. The method of claim 34, wherein said cell culture comprising porcine primordial germ cells contains undifferentiated cultured cells from an undifferentiated porcine primordial germ cell-derived cell line.

40. The method of claim 34, wherein said transgenic pig is generated by a method comprising:

(a) injecting said undifferentiated porcine primordial germ cells that contain said selected DNA segment into a blastocyst from a pig;

(b) transferring said blastocyst into a synchronized recipient female pig to produce a pregnant pig; and

(c) allowing gestation in said pregnant pig to proceed for a period of time sufficient to allow the development of a viable transgenic pig.

41. The method of claim 34, wherein said transgenic pig is generated by a method comprising:

(a) isolating a nucleus from said undifferentiated porcine primordial germ cells that contain said selected DNA segment and injecting said nucleus into an enucleated oocyte from a pig;

(b) transferring said oocyte into a synchronized recipient female pig to produce a pregnant pig; and

(c) allowing gestation in said pregnant pig to proceed for a period of time sufficient to allow the development of a viable transgenic pig.

42. The method of claim 34, wherein said transgenic pig is generated by a method comprising:

(a) aggregating said undifferentiated porcine primordial germ cells that contain said selected DNA segment with an early stage embryo of a pig;

(b) transferring said embryo into a synchronized recipient female pig to produce a pregnant pig; and

(c) allowing gestation in said pregnant pig to proceed for a period of time sufficient to allow the development of a viable transgenic pig.

43. The method of claim 34, wherein said selected DNA segment comprises at least a first coding region that encodes a protein selected from the group consisting of an interleukin, collagen, interferon, blood protein, hormone, growth factor, cytokine, enzyme, receptor, binding protein, immune system protein, antigen, muscle protein and an oncogene receptor.

44. The method of claim 43, wherein said selected DNA segment comprises at least a first coding region that encodes a protein selected from the group consisting of SREHP, GP63, actinobacillus, pleuropneumoniae, pseudomonas aeruynosa, OprF, myelin basic protein, insulin, hCD59, DAF (CD55), factor IX, urokinase, .alpha.-antitrypsin, tissue plasminogen activator, protein C, activin, adenosine deaminase, angiotensinogen I, antithrombin III, alpha I antitrypsin, apolipoprotein A-I, apolipoprotein A-II, apolipoprotein C-I, apolipoprotein C-II, apolipoprotein C-III, apolipoprotein E, atrial natriuretic factor, chorionic gonadotropin, alpha chain, beta chain, pro (rennin) chymosin, factor B complement, complement C2, complement C3, complement C4, complement C9, corticotropin releasing factor, epidermal growth factor, c-erb B, epoxide dehydratase, erythropoietin, C1 esterase inhibitor, factor VIII, factor IX, Christmas factor, factor X, fibrinogen A alpha, gamma B beta, gastrin releasing peptide, prepro glucagon, growth hormone, RF growth hormone, somatocrinin, hemopexin, inhibin, prepro insulin, insulin-like growth factor I, insulin-like growth factor II, alpha interferon, multiple leukocyte, fibroblast beta interferon, gamma interferon, interleukin-1, T-cell interleukin-2, growth factor, interleukin-3, two forms kininogen, beta subunit leuteinizing hormone, leuteinizing hormone, releasing hormone, lymphotoxin, mast cell growth factor, beta subunit nerve growth factor, PGDF c-sis oncogene, chain A, pancreatic polypeptide, icosapeptide, parathyroid hormone, prepro plasminogen, plasminogen activator, prolactin, proopiomelanocortin, protein C, prothrombin, relaxin, prepro renin, somatostatin, prepro tachykinin, substance P, substance K, urokinase and prepro vasoactive intestinal peptide protein.

45. A method of preparing a porcine blastocyst that contains a selected DNA segment, comprising:

(a) introducing said selected DNA segment into a cell culture comprising porcine primordial germ cells to obtain candidate porcine primordial germ cells that contain said selected DNA segment;

(b) plating said candidate porcine primordial germ cells that contain said selected DNA segment on feeder cells, said feeder cells at a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 an about 10.sup.6 cells/cm.sup.2, in a culture medium comprising an effective amount of basic fibroblast growth factor and an apoptosis inhibitor, to obtain undifferentiated porcine primordial germ cells that contain said selected DNA

segment; and

(c) injecting said undifferentiated porcine primordial germ cells that contain said selected DNA segment into a porcine blastocyst, thereby preparing a porcine blastocyst that contains said selected DNA segment.

46. A method of preparing a porcine oocyte that contains a selected DNA segment comprising:

(a) introducing said selected DNA segment into a cell culture comprising porcine primordial germ cells to obtain candidate porcine primordial germ cells that contain said selected DNA segment;

(b) plating said candidate porcine primordial germ cells that contain said selected DNA segment on feeder cells, said feeder cells at a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 cells/cm.sup.2, in a culture medium comprising an effective of basic fibroblast growth factor and an apoptosis inhibitor, to obtain undifferentiated porcine primordial germ cells that contain said selected DNA segment;

(c) isolating a nucleus from said undifferentiated porcine primordial germ cells that contain said selected DNA segment; and

(d) injecting said nucleus into an enucleated porcine oocyte, thereby preparing a porcine oocyte that contains said selected DNA segment.

47. (Amended) A method of preparing an early stage porcine embryo that contains a selected DNA segment, comprising:

(a) introducing a selected DNA segment into a cell culture comprising porcine primordial germ cells to obtain candidate porcine primordial germ cells that contain said selected. DNA segment;

(b) plating said candidate porcine primordial germ cells that contain said selected DNA segment on feeder cells, said feeder cells at a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 cells/cm.sup.2, in a culture medium comprising an effective amount of basic fibroblast growth factor and an apoptosis inhibitor, to obtain undifferentiated porcine primordial germ cells that contain said selected DNA segment; and

(c) aggregating said undifferentiated porcine primordial germ cells that contain said selected DNA segment with an early stage porcine embryo, thereby preparing an early stage porcine embryo that contains said selected DNA segment.

48. A cell culture comprising:

(a) porcine primordial germ cells;

(b) feeder cells sufficient to achieve a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 feeder cells/cm.sup.2 ; and

(c) an mount of basic fibroblast growth factor and an apoptosis inhibitor

effective to promote the growth and continued proliferation of said porcine primordial germ cells.

49. The cell culture of claim 48, wherein said apoptosis inhibitor is a serine protease apoptosis inhibitor.

50. The cell culture of claim 48, wherein said apoptosis inhibitor is an antioxidant apoptosis inhibitor.

51. The cell culture of claim 48, wherein said apoptosis inhibitor is selected from the group consisting of bcl-2, Bcl-x1, Mcl-1, Bak, A1, A20, TNF-.alpha., nerve growth factor, epidermal growth factor, insulin-like growth factor-I, insulin-like growth factor-I receptor, 3-aminobenzamide, aphidocolin, L-ascorbic acid, catalase, follicle stimulating hormone, vasoactive intestinal peptide, cyclic GMP, hCG, interleukin-1.beta., superoxide dismutase, aurintricarboxylic acid, BAPTA/AM, caffeine, calpain inhibitor I, calpain inhibitor II, leupeptin, N-ethyl maleimide, cyclosporin A, acetyl-leucyl-leucyl-normethional, 3,4-dichloroisocoumarin, diisopropylfluorophosphate, phenylmethylsulfonyl fluoride, tetrodotoxin, nimodipine, verapamil, flunarizine, R56865, forskolin, genistein, herbimycin A, interleukin-6, calyculin A, okadaic acid, calyculin A, phorbol-12-myristate-13-acetate, 1-Pyrrolidinecarbodithioic acid, nifedipine, nisoldipine, aurintricarboxylic acid, spermine, thioredoxin, N.sup..alpha. -Tosyl-L-Phe chloromethyl ketone and N.sup..alpha. -Tosyl-L-Lys chloromethyl ketone.

52. The cell culture of claim 51, wherein said apoptosis inhibitor is N-Acetyl-L-cysteine.

53. The cell culture of claim 48, wherein said porcine primordial germ cells comprise at least a first exogenous DNA segment.

54. A kit comprising, in suitable container means:

(a) porcine primordial germ cells;

(b) feeder cells sufficient to achieve a density of between about 2.5.times.10.sup.5 cells/cm.sup.2 and about 10.sup.6 feeder cells/cm.sup.2 ; and

(c) an amount of basic fibroblast growth factor and an apoptosis inhibitor effective to promote the growth and continued proliferation of said porcine primordial germ cells.

55. The kit of claim 54, wherein said apoptosis inhibitor is a serine protease apoptosis inhibitor.

56. The kit of claim 54, wherein said apoptosis inhibitor is an antioxidant apoptosis inhibitor.

57. The kit of claim 54, wherein said apoptosis inhibitor is selected from the group consisting of bcl-2, Bcl-x1, Mcl-1, Bak, A1, A20, TNF-A, nerve growth factor, epidermal growth factor, insulin-like growth factor-I, insulin-like growth factor-I receptor, 3-aminobenzamide, aphidocolin, L-ascorbic acid, catalase, follicle stimulating hormone, vasoactive intestinal peptide, cyclic GMP, hCG, interleukin-1.beta.,superoxide dismutase, aurintricarboxylic acid, BAPTA/AM, caffeine, calpain inhibitor I, calpain inhibitor II, leupeptin, N-ethyl maleimide, cyclosporin A, acetyl-leucyl-leucyl-normethional, 3,4-

dichloroisocoumarin, diisopropylfluorophosphate, phenylmethylsulfonyl fluoride, tetrodotoxin, nimodipine, verapamil, flunarizine, R56865, forskolin, genistein, herbimycin A, interleukin-6, calyculin A, okadaic acid, calyculin A, phorbol-12-myristate-13-acetate, 1-Pyrrolidinecarbodithioic acid, nifedipine, nisoldipine, aurintricarboxylic acid, spermine, thioredoxin, N.sup..alpha. -Tosyl-L-Phe chloromethyl ketone and N.sup..alpha. -Tosyl-L-Lys chloromethyl ketone.

58. The kit of claim 57, wherein said apoptosis inhibitor is N-Acetyl-L-cysteine.

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File 155:MEDLINE(R) 1951-2005/May W4
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E3      3563     1 *THIOREDOXIN
E4         5        THIOREDOXIN --ADMINISTRATION AND DOSAGE --AD
E5         2        THIOREDOXIN --ANALOGS AND DERIVATIVES --AA
E6        90        THIOREDOXIN --ANALYSIS --AN
E7        41        THIOREDOXIN --ANTAGONISTS AND INHIBITORS --AI
E8       117        THIOREDOXIN --BIOSYNTHESIS --BI
E9        53        THIOREDOXIN --BLOOD --BL
E10        3        THIOREDOXIN --CHEMICAL SYNTHESIS --CS
E11      517        THIOREDOXIN --CHEMISTRY --CH
E12       11        THIOREDOXIN --CLASSIFICATION --CL

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E14      634  THIOREDOXIN --GENETICS --GE
E15       34  THIOREDOXIN --IMMUNOLOGY --IM
E16      109  THIOREDOXIN --ISOLATION AND PURIFICATION --IP
E17     1016  THIOREDOXIN --METABOLISM --ME
E18        1  THIOREDOXIN --PHARMACOKINETICS --PK
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E20        9  THIOREDOXIN --PHYSIOLOGY --PH
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E22       15  THIOREDOXIN --SECRETION --SE
E23       10  THIOREDOXIN --THERAPEUTIC USE --TU
E24        2  THIOREDOXIN --ULTRASTRUCTURE --UL

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             9  THIOREDOXIN --PHYSIOLOGY --PH
            10  THIOREDOXIN --THERAPEUTIC USE --TU
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                --TU'
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E28       36        THIOREDOXIN F
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E30       23        THIOREDOXIN H
E31        6        THIOREDOXIN H2 PROTEIN, PLANT
E32      657     2  THIOREDOXIN REDUCTASE (NADPH)
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#5 **Related Articles** for **PubMed** (Select **10097175**)	14:46:09	1
#1 Search **thioredoxin therapy**	14:42:51	1

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L3: Entry 9 of 18 File: USPT Jun 18, 2002

DOCUMENT-IDENTIFIER: US 6406701 B1
TITLE: Method and compositions for preventing or reducing HIV infection

Detailed Description Text (77):
These observations, together with the uncompetitive inhibitory effect of thioredoxin on LAPase raise the possibility of the use of combined therapy for the prevention and treatment of AIDS.

CLAIMS:

3. The method according to claim 2 wherein the thiol containing compound is thioredoxin.

9. The method according to claim 8 wherein the thiol containing compound is selected from the group consisting of thioredoxin and glutathione.

19. The method according to claim 18 wherein the thiol containing compound is thioredoxin.

24. The method according to claim 23 wherein the thiol containing compound is thioredoxin.

Previous Doc Next Doc Go to Doc#

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SYSTEM:OS  - DIALOG OneSearch
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         2001 (c) Action Potential
  File  94:JICST-EPlus 1985-2005/Mar W4
         (c)2005 Japan Science and Tech Corp(JST)
  File  98:General Sci Abs/Full-Text 1984-2004/Dec
         (c) 2005 The HW Wilson Co.
  File 135:NewsRx Weekly Reports 1995-2005/May W3
         (c) 2005 NewsRx
*File 135: New newsletters are now added. See Help News135 for the
complete list of newsletters.
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  File 149:TGG Health&Wellness DB(SM) 1976-2005/May W3
         (c) 2005 The Gale Group
  File 156:ToxFile 1965-2005/May W3
         (c) format only 2005 The Dialog Corporation
*File 156: ToxFile has been reloaded with the 2005 MeSH.
Please see HELP NEWS 156 for details.
  File 159:Cancerlit 1975-2002/Oct
         (c) format only 2002 Dialog Corporation
*File 159: Cancerlit is no longer updating.
Please see HELP NEWS159.
  File 162:Global Health 1983-2005/Mar
         (c) 2005 CAB International
  File 164:Allied & Complementary Medicine 1984-2005/May
         (c) 2005 BLHCIS
  File 172:EMBASE Alert 2005/May W2
         (c) 2005 Elsevier Science B.V.
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information.
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         (c) 2005 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
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              OR 'THIOREDOXIN --THERAPEUTIC USE --TU'
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S3         23   S1 NOT S2
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              AL DRUG ADMINISTRATION'
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S8         18   S7/2001:2005
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